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CERTAIN ASPECTS CONCERNING OXYGEN METABOLISM OF THE BODY EXPOSED TO PROLONGED ACCELERATIONS

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CERTAIN ASPECTS CONCERNING OXYGEN METABOLISM OF THE BODY EXPOSED TO PROLONGED ACCELERATIONS

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ABSTRACT. Changes in oxygen metabolism of muscular and brain tissues of animals exposed to prolonged transverse accelerations (oxygen tension and redox potential) are described. The paper also discusses the total oxygen consumption and body temperature under the same conditions. A many-faceted approach to the processes studied allows us to establish new regularities in oxygen metabolism of muscular and brain tissues. Their evaluation helps to understand the role of the changes in tolerance of the body for accelerations on the whole.

In this study, we attempted to evaluate the interrelation of a number of indices of the oxygen metabolism in a complex form of hypoxia induced by the action of prolonged transverse accelerations. The state of the oxidative processes in the tissues (brain and skeletal musculature) was evaluated on the basis of the following mutually supplementary parameters: oxygen pressure, redox potential, muscle temperature, total oxygen consumption of the body, and body temperature. It is well known that the oxygen tension (PO_2) gives an idea of the condition under which the oxidative processes take place, that it constitutes the resultant of many factors and that, in the final analysis, it reflects the correlation between the rate of oxygen diffusion from the blood into the tissue fluids and the rate of oxygen binding (Ye.A.Kovalenko and associates). However, the data on the dynamics of oxygen tension in the tissues are insufficient for a definite judgement as to its utilization in the biological oxidation processes.

More direct information on the level of oxidative processes in experiments in vivo is given by the redox potential (Eh) to which particular significance was attached in the present study.

An interpretation of the value of the redox potential of a system as complex as the living biological substrate is an exceedingly difficult problem, which has not yet been completely solved. This is due to the fact that the living body is not a closed system but a thermodynamically open one which is in a stationary state and where fluxes of energy and matter in the direction organism-medium and medium-organism are not interrupted. Furthermore, it is not a question of separate and isolated redox reactions taking place in the body /27 but rather of a complex system of such reactions, where the process of oxidation-reduction itself is irreversible. Thus, it is natural that the redox potential, measured under the conditions of such an open system, should have a somewhat different meaning than in a closed equilibrium system.

* Numbers in the margin indicate pagination in the foreign text.

According to present ideas (Cater; M.Ye.Rayskina and B.M.Shargorodskiy), the redox potential measured in an open system expresses the instantaneous resultant of the kinetics of irreversible processes under flow conditions. The stationary nature of the Eh is determined by the constancy of the concentrations of the oxidative and reductive forms of the reversible pairs, by the concentrations of the irreversible reducers, the content of free oxygen, the velocities of the biochemical reactions, and the diffusion of metabolites. Naturally many actions on the body (introduction of certain substances, change in composition of the inhaled atmosphere, etc.) may disturb the stationary state and change the value of the total redox potential. In this case, an increase in the Eh indicates the relative predominance of the oxidative processes and the accumulation of oxidized forms in the system; a decrease of this index, instead, reflects the relative suppression of the oxidative processes and the accumulation in the medium of reduced (incompletely oxidized) products.

These data acquire the greatest interest when we consider them in a group together with other indices of the oxygen regime of the body. Such an approach naturally gives more grounds for evaluation of the relation between the supply of oxygen to the tissues and its consumption in all stages of hypoxia.

Our experiments were performed on white rats of the Wistar strain, weighing 150 - 180 g. The prolonged accelerations of 10 and 25 g of 6 min in duration were produced on a centrifuge with an arm of 1.8 m length. The experimental animal was placed in a special profiled box attached to the rotary frame of the centrifuge. The box consisted of a removable nonrotating stage made of organic glass and wax.

The oxygen tension in the brain (subcortical layer) and in the triceps surae were determined in the acute experiment by the polarographic method, using open-type electrodes. The platinum electrodes for recording the PO_2 in the muscle tissue was in the form of a needle of 20 mm length and 0.3 mm diameter, insulated by epoxide resin along its entire length except for the working part. The electrode for measuring the PO_2 in the brain tissue was a needle of 3 mm length, enclosed in a holder of organic glass for attachment to the skull. The anode was a silver chloride electrode or a saturated calomel half-cell, connected with the animal by a flexible potassium chloride agar siphon. The anode was introduced into the skin at a distance of 3 mm from the cathode. The oxygen tension was registered continuously throughout the experiment on a recording polarograph of the "Selector" type manufactured by the "Atlas Werke". The PO_2 was measured in percent of its initial level. As 100%, we used the PO_2 recorded while breathing air before the beginning of the experiment.

The redox potential of the tissues was determined potentiometrically by measuring the emf of a circuit consisting of an indicator electrode (platinum) and a reference electrode (calomel). The former here played the role of an electron donor or acceptor, depending on whether reducing or oxidizing processes predominated in the system. The instrument was calibrated by the method described by B.M.Shargorodskiy and B.P.Rastorguyev. The reference electrode used was a saturated calomel half-cell with a self-potential of +200 mv. The design of the electrode for measuring the Eh did not differ from that of the electrodes used for recording the oxygen tension. The redox potential was measured by a pH-potentiometer of the TA-53 type made by the Godart Company,

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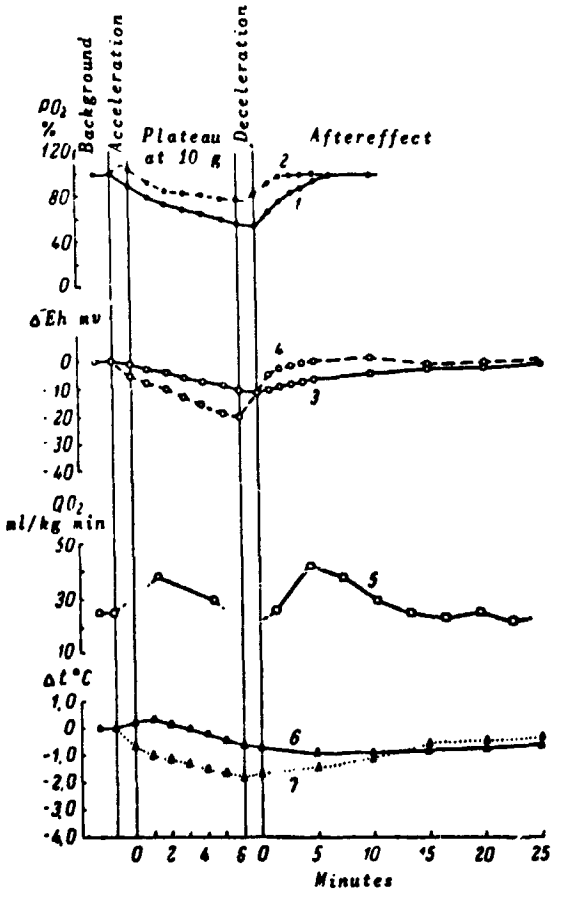

Fig.1 Dynamics of Variations of PO_2. (1 - in muscle; 2 - in brain). Eh (3 - in muscle and 4 - in brain, 5 - QO_2 and temperature, 6 - in the muscle, 7 - rectal) under the action of a 10 g acceleration.

with an accuracy of ±1 mv.

The tissue temperature was measured by needle thermoresistors, and the body temperature (rectal) by a special thermocouple on an electric thermometer of type TE-5, made by the Ellab Company. Its accuracy was ±0.05°C.

The level of total oxygen consumption by the body was determined by a manometer. The value of the QO_2 (reduced + normal conditions) over 3 min intervals was calculated from the pressure variation in the hermetically sealed chamber which contained the experimental animal. The water vapor and carbon dioxide liberated was removed from the system by suitable absorbers.

Ninety control tests and 260 acceleration experiments were performed. The numerical material was statistically processed according to the scheme of the classical analysis for the confidence level of 0.95 generally used in medicobiological research. The character of the distribution of the indices studied was parametrically determined, and the mathematical expectation (arithmetic mean), the variance, and the standard deviation were calculated. The reliability of the difference between the mean-square values was established by the aid of the Student criterion. To obtain the mean dynamics of the indices studied during the action of the acceleration, the averaging method generally adopted in the theory of random functions was used (with the intervals between the sections being 1 min). In this case, the initial data made use of the differences between the current and initial values of the parameter.

The calculations were performed on an NAIRI digital computer. So as to keep the detail on the graphs to a minimum (Figs.1 and 2), only the values of the mathematical expectations are shown. The differences mentioned in the text are statistically reliable.

As expected, a greater acceleration (25 g) led to more extensive changes in the oxygen regime of the body. For example, the reduction in both Eh and PO_2 in brain and muscle by the end of the 25 g acceleration period was almost twice as extensive as in experiments with a 10 g acceleration. The same is true of the accumulation of the total oxygen debt, which was judged from the increase

in oxygen consumption (QO_2) during the aftereffect period. The total oxygen debt in the experiments at 10 g was 120 ml/kg, while in the 25 g experiments it was 290 ml/kg.

It is well known that the compensatory reactions of the organism during prolonged action of accelerations, as in many other extremal influences and pathological states, are primarily directed toward maintenance of normal vital activity of the main functional systems and organs, above all of the brain. Thus, it has been found (A.S.Barer et al.) that a purposeful selective compensatory reaction exists, which maintains blood circulation specifically in the cerebral vessels. Disturbances of circulation in the external vessels of the head, in these cases, started at considerably smaller accelerations.

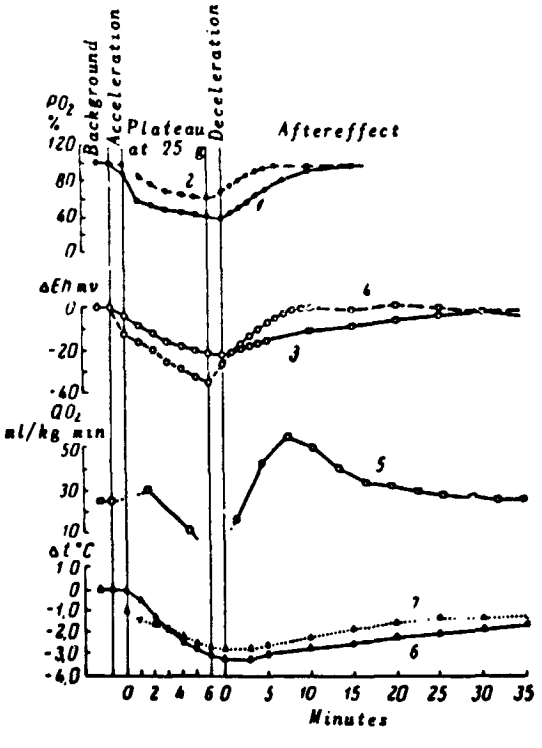

Fig.2 Dynamics of Variations of PO_2. (1 - in the muscle, 2 - in the brain), Eh (3 - in the muscle, 4 - in the brain, 5 - QO_2 and temperature, 6 - in the muscle, 7 - rectal) under the action of a 25 g acceleration.

In our experiments this proposition was confirmed by maintenance of a higher PO_2 level in the brain tissue than in the skeletal musculature. However, judging from the variation in the redox potential, which directly illustrates the state of the oxidative processes in the tissues, it may be said that, already at 10 g acceleration, the organism is no longer able to adequately supply oxygen to the brain tissues. Thus, despite the higher PO_2 level, the shift in oxidative equilibrium in the brain is deeper than in the muscular tissue. In evaluating this phenomenon, one must take account of the fact that the intensity of the redox processes is highest in the brain tissue (G.Ye.Vladimirov and N.S.Panteleyeva, V.S.Shapot), so that even a slight decline in oxygen supply will lead to an accumulation of a large quantity of incompletely oxidized products.

Our results confirm once again the proposition that the primary limiting factor in the tolerance of prolonged accelerations by the body is not so much the accumulation of the total oxygen debt but the disturbance of the oxygen metabolism in vital organs and specifically in the brain (A.S.Barer et al.).

Let us now discuss the course of development of these processes. The sensitivity of the body to the group of phenomena accompanying the action of transverse accelerations indicates that the disturbance of stationarity of the indices of oxygen metabolism already takes place during the "acceleration" period of the centrifuge, which in our experiments lasted 60 sec. Here we observe both a decrease in oxygen tension of the tissues and a negative shift of

the redox equilibrium. In this connection, our attention is struck by the brief rise in tissue PO_2 at low accelerations (Fig.1), indicating the rapid purposive switch-on of the compensatory reactions to the body. As already noted, the degree of compensation, however, became insufficient on further intensification of the force of action.

During the entire six-minute period of the "plateau", the manifestations of hypoxia in the brain and muscular tissues continue to deepen. During this same time there begins a progressive decline in body temperature, exactly as the level of oxygen consumption of the body continues to decline at pronounced anoxia of the tissues. The latter phenomenon may to some extent be connected with the inadequacy of pulmonary ventilation, due to difficulties in the biomechanics of respiration and the disturbance of the gas exchange in the alveolocapillary connection on account of shifts in the lesser circulation (A.S.Barer et al.; G.A.Golov; A.A.Kiselev; Nolan et al.).

During the aftereffect, the initial stationary state which had been disturbed by the acceleration is again restored.

The fact that the restoration of oxidative equilibrium in the brain tissue is more intense and ends at earlier periods than, for instance, in the skeletal musculature, is interesting and, from the viewpoint of biological expediency, understandable enough. In this case, normalization of the PO_2 takes place considerably before normalization of the redox potential and of the total oxygen consumption of the body.

Thus, in experiments with an acceleration of 25 g, the PO_2 of the muscular tissue is normalized in the 10 - 15^{th} minute after the action, while the Eh and QO_2 are normalized almost simultaneously in the 30 - 35^{th} minute of this period.

Consequently, normalization of the PO_2 does not yet in itself mean elimination of the state of hypoxia or of the total oxygen deficiency in the body. Our experiments likewise permit the conclusion that the total oxygen deficiency is determined primarily by the degree of defiency in the skeletal musculature, whose mass is of dominant importance in the body.

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